"MANAGEMENT OF SECRETORY OTITIS MEDIA – A COMPARATIVE STUDY"

Submitted in partial fulfillment of the requirements for

M.S. DEGREE BRANCH -IV OTORHINOLARYNGOLOGY

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DECLARATION

I solemnly declare that the dissertation entitled "MANAGEMENT OF SECRETORY OTITIS MEDIA –A COMPARATIVE STUDY" is done by me at the Coimbatore Medical College Hospital, Coimbatore during 2007-2009 under the guidance and supervision of Prof.K.B.MOTHILAL , M.S., D.L.O.

This dissertation is submitted to The Tamilnadu Dr. M.G.R Medical University, towards partial fulfillment of regulation for the award of M.S. DEGREE (BRANCH–IV) in Otorhinolaryngology.

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CERTIFICATE

This is to certify that this dissertation entitled "**MANAGEMENT OF SECRETORY OTITIS MEDIA-A COMPARATIVE STUDY** " submitted by **Dr. D.Y.RAJ PRAKASH,** appearing for M.S. (E.N.T) Degree (Branch IV) examination in March 2010 is a bonafide record of work done by him under my guidance and supervision in partial fulfillment of regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai. I forward this to the Tamil Nadu Dr.M.G.R. Medical University, Chennai, Tamil Nadu, India.

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CONTENTS

INTRODUCTION

Secretory otitis media is the most common cause of hearing impairment in children. It is defined as the persistence of serous or mucoid middle ear effusion for 12 weeks or more.[1] It is also called as otitis media with effusion, catarrhal otitis media, exudative otitis media, seromucinous otitis media, non-suppurative otitis media. The term secretory is appropriate as it reflects a particular aspect of pathological process. The term otitis media with effusion allows differentiation of the type of effusion and facilitates distinction into serous and mucinous, acute and chronic forms.

In many children,secretory otitis media is preceded by an episode of acute otitis media.This is true especially in younger children because of the higher incidence of upper respiratory tract infection. Acute otitis media is usually triggered by a viral respiratory tract infection which damages the epithelium of Eustachian tube, resulting in retention of middle ear fluid. Hence secretory otitis media will be present temporarily in many children after an episode of acute otitis media.

Secretory otitis media is generally self-limiting, but may occur during a period when poor hearing will impede speech and language development. The effects are mostly short term, but in children in whom the condition recurs throughout childhood, some effects on behaviour and cognition are detectable up to the age of 10 years and beyond.[2] In adults it can lead on to atelectatic otitis media which can sometimes lead on to cholesteatoma formation. This study was performed to clinically evaluate and to find out the effective method of managing this condition. This study was performed in Department of ENT, Coimbatore Medical College & Hospital, Coimbatore in patients who report with symptoms of conductive hearing loss.

OBJECTIVES:

1) To identify the distribution of age and sex in cases of secretory otitis media.
2) To find out the commonest predisposing factor.
3) To evaluate the different clinical parameters of secretory otitis media.
4) To compare the efficacy of medical management with various surgical procedures.
5) To arrive at the best treatment modality and to compare with the published literature.

HISTORY

The problem of fluid in middle ear has been recognised for hundred of years. In 1869, Politzer described a condition that he termed ‘otitis media catarrhalis’in his classic book ‘The diseases of ear’. He recognised secretory and adhesive forms of the condition. The treatment that he advocated consisted of insufflation of air and paracentesis of the middle ear, which were intended to equalize atmospheric pressure on both sides of tympanic membrane. The principles of both ventilation and drainage of middle ear have remained the same in the management of middle ear effusion till date. This received little attention till second world war.

The clinical challenge of diagnosing this disorder was described by Hooper (1950). He elucidated the various clinical features of middle ear effusion.

An important contribution to the management of middle ear effusion was introduced by Armstrong in 1954 when he advocated the use of an indwelling polyethylene tube thro the the tympanic membrane to achieve ventilation and drainage,as described by Politzer.

EPIDEMIOLOGY:

The epidemiology of Secretory otitis media has been studied in several countries like Belgium, Holland , Denmark, Spain, U.K, U.S.A, and India.

PREVALANCE:

The prevalence of a condition is the proportion of a population that has a condition at any one time. It is an indicator of potential clinical workload. In children the main determinants are age of the child and season of the year.

AGE OF THE CHILD:

Secretory otitis media shows a bimodal prevelance with first peak at 2 year when the child attends a playgroup or nursery school, second peak at around 5years of age when most child attend a primary school.[3] By the age of 7or 8 years, prevelance falls.

SEASON OF THE YEAR:

The effect of season on the prevelance of secretory otitis media is well recognised in temperate climate with twice the children diagnosed in winter as compared to summer months. [4,5,6, 7] This is mainly due to the increased incidence of upper respiratory tract infection in winter and

greater chances of passing them among children in winter.The prevelance of secretory otitis media in Mediterranean[8,9] and subtropical countries [10,11] does not appear to be different overall from those in temperate countries.

HEREDITARY FACTORS :

In a same sex twin /triplet prospective cohort study Casselbrant et al [12] look at sets where zygosity was known. In children who had OME during the first two years of life, there was greater concordance in monozygotic twins in the number and duration than in dizygotic twins.

RACE:

A study by paradise et al showed that the prevalence of OME in black children was no different from white, provided factors like socioeconomic group and child contacts are controlled.[13] At present there is insufficient evidence to examine any effect on race, apart from black and white where no difference has been shown.

GENDER:

Some multivariate studies report that there is no difference in boys and girls. In some study more risk in boys[14,15] and others in girls.[16]

BREAST FEEDING:

In numerous studies, breast-feeding has been reported as reducing the risk of ear and respiratory infections, and hence, of OME . Maternal immunity has been proposed as the mechanism underlying this protective effect. Breast-fed infants carry reduced numbers of bacteria in their nasopharynx. A two-fold increase in risk of first episodes of AOM or OME was found in infants exclusively formula-fed as opposed to those breast fed for 6 months.

However, a large longitudinal birth cohort study of over 12,000 children born in 1970, whilst confirming other established risk factors, found the protective effect of breast-feeding to be weak.[17] This illustrates that depending on the nature of the population, the strength of the risk factors is variable.

DAY CARE:

Attending day-care centres increases the risk of OME up to three times than for children cared for at home or by a child-care giver in a small group. The risk is also higher in families with a large number of siblings at home. The influences of season, day-care attendance and family size are likely to be inter-related variables, the common element being increased exposure to both viral and bacterial respiratory pathogens.

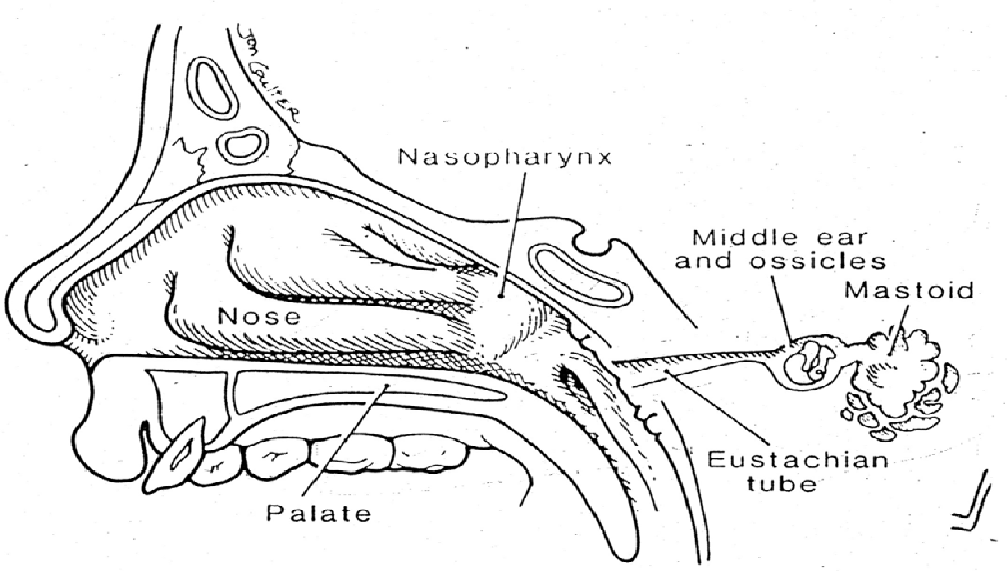
PARENTAL SMOKING:

Maternal, but not paternal smoking was shown to be associated with presence of middle ear effusion in the children (8–18 years) of British servicemen . In a large, longitudinal cohort study, a similar dependence on parent gender was found, suggesting a direct relation to dose[18] . Controlling statistically for further influential factors, a systematic quantitative review from the UK (Strachan and Cook 1998) concluded that there is likely to be a causal relationship between parental smoking and both acute and chronic middle ear disease in children.

However in multivariate analysis when other factors have been controlled, no effect of parental smoking is detected [19] or it is present for smokers of up to 20 cigarettes per day but the risk decreases if it is more than 20 cigarettes per day.[20]

ANATOMY OF THE EUSTACHIAN TUBE & MIDDLE EAR CLEFT:

The Eustachian tube, middle ear cavity proper and the aditus with antrum, mastoid air cell system form the middle ear cleft. The eustachian tube lumen is wider at both the proximal (nasopharyngeal) and distal (middle ear) ends than in the midportion. The isthmus is the narrowest party of the eustachian tube.On the lateral wall of the nasopharynx, a prominence, the torus tubarius, protrudes into the nasopharynx. This prominence is formed by the abundant soft tissue overlying the cartilage of the eustachian tube. Anterior to this is the triangular nasopharyngeal orifice of the tube. From the torus, a raised ridge of mucous membrane, the salpingopharyngeal fold, descends vertically. On the posterior wall of the nasopharynx lie the adenoids, or pharyngeal tonsil, composed of abundant lymphoid tissue. Above the tonsil is a variable depression within the mucous membrane called the pharyngeal bursa. Behind the torus lies a deep pocket, extending to the nasopharynx posteriorly along the medial border of the eustachian tube. This pocket, the fossa of Rosenmuller, varies in height from 8 to 10 mm and in depth from 3 to 10 mm. Adenoid tissue usually extends into this pocket, giving soft-tissue support to the tube.



Nasopharynx & middle ear cleft

In the adult, the eustachian tube is longer than that in the infant and young child. Most of the increase in length takes place before age 6 years. The eustachian tube has been reported to be as short as 30 mm and as long as 40 mm, but the usual range of length reported in the literature is 31 to 38 mm . The posterior third (11 to 14 mm) of the adult tube is osseous, and the anterior two-thirds (20 to 25 mm) is composed of membrane and cartilage . In adults, the eustachian tube lies at an angle of 45 degrees in relation to the horizontal plane. In infants, this inclination is only 10 degrees. The osseous eustachian tube (protympanum) lies completely within the petrous portion of the temporal bone and is directly continuous with the anterior wall of the superior portion of the middle

ear. The junction of the osseous tube and the epitympanum lies 4 mm above the floor of the tympanic cavity.

The osseous (protympanic or middle-ear) portion of the tube has a course that is linear anteromedially, following the petrous apex and deviating little from the horizontal plane. The lumen is roughly triangular, measuring 2 to 3 mm vertically and 3 to 4 mm along the horizontal base. The healthy osseous portion is open at all times, in contrast to the fibrocartilaginous portion, which is closed at rest and opens during swallowing or when forced open, such as during the Valsalva manoeuvre. The osseous and cartilaginous portions of the eustachian tube meet at an irregular bony surface and form an angle of about 160 degrees with each other. The medial wall of the bony portion of the eustachian tube consists of two parts, posterior (labyrinthine) and anterior (carotid) whose size, shape, and relation depend on the position of the internal carotid artery. The average thickness of the anteromedial portion is 1.5 to 3 mm, and in 2% of persons, the wall is absent, exposing the carotid artery.

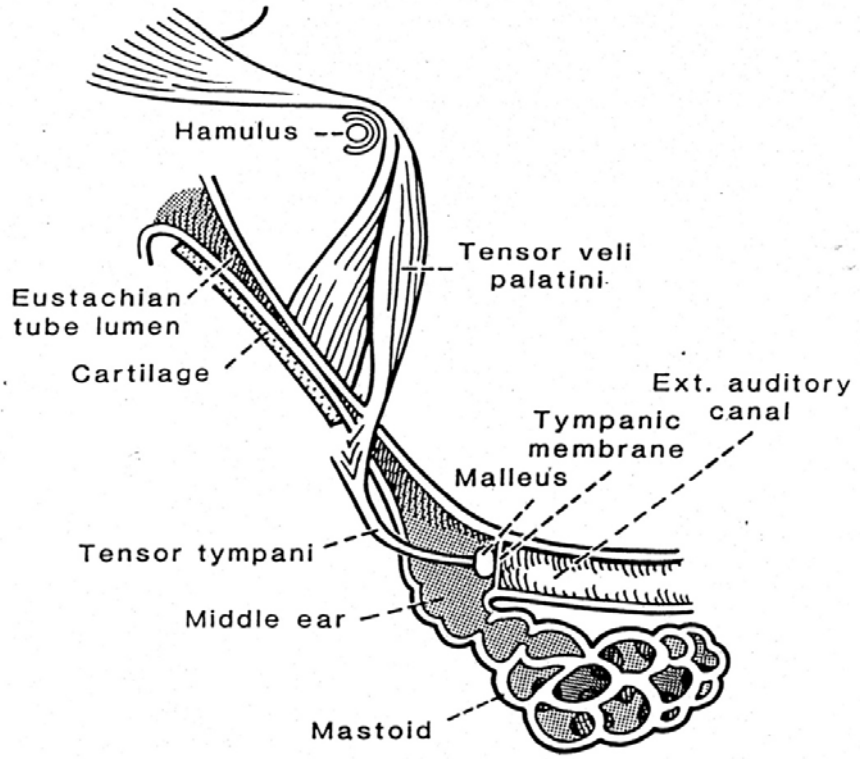
The cartilaginous tube then courses anteromedially and inferiorly, angled in most cases 30 to 40 degrees to the transverse plane and 45 degrees to the sagittal plane. The tube is applied closely to the basal aspect of the skull and fitted to the sulcus tubae between the greater wing of the sphenoid bone and the petrous portion of the temporal bone. The

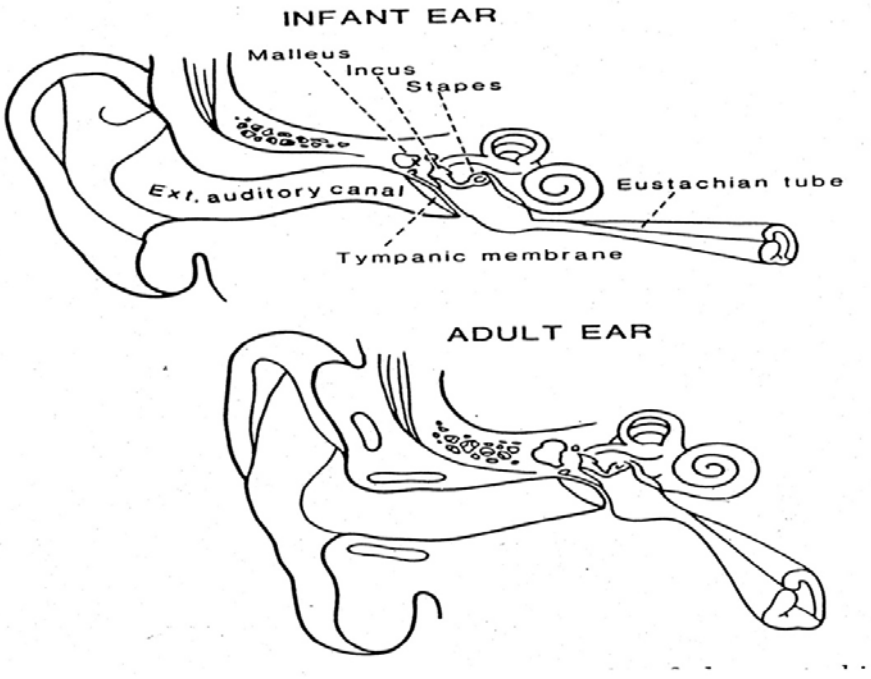
cartilaginous tube is attached firmly at its posterior end to the osseous orifice by fibrous bands and usually extends some distance (3 mm) into the osseous portion of the tube. At its inferomedial end, it is attached to a tubercle on the posterior edge of the medial pterygoid lamina .

The tube in its cartilaginous portion has a crook-shaped mediolateral superior wall . It is completed laterally and inferiorly by a veiled membrane that serves as the site of attachment of the fibers of the dilator tubae, or tensor veli palatini muscle . The tubal lumen is shaped like two cones joined at their apexes. The junction of the cones is the narrowest point of the lumen and has been called the isthmus. Its position is usually described as at or near the junction of the osseous and cartilaginous portions of the tube. The lumen at this point is about 2 mm high and 1 mm wide . From the isthmus, the lumen expands to about 8 to 10 mm in height and 1 to 2 mm in diameter at the pharyngeal orifice. Tubal cartilage increases in mass from birth to puberty, and this development has physiologic implications. The cartilaginous eustachian tube does not follow a straight course in the adult but extends along a curve from the junction of the osseous and cartilaginous portions to the medial pterygoid plate, approximating the cranial base for the greater part of its course. The eustachian tube crosses the superior border of the superior constrictor muscle immediately posterior to its terminus within the nasopharynx. The thickened anterior fibrous investment of the medial

cartilage of the tube presses against the pharyngeal wall to form a prominent fold, the torus tubarius, which measures 10 to 15 mm thick . The torus is the site of origin of the salpingopalatine muscle and is the point of origin of the salpingopharyngeal muscle, which lies within the inferoposteriorly directed salpingopharyngeal fold .

The mucosal lining of the eustachian tube is continuous with that of the nasopharynx and middle ear and is characterized as respiratory epithelium. Structural differentiation of this mucosal lining is evident wherein mucous glands predominate at the nasopharyngeal orifice, and graded change occurs to a mixture of goblet, columnar, and ciliated cells near the tympanum. The lining is folded, which provides greater surface area . Mucosa associated lymphoid tissue also is present .


Hamulus
Tensor veli palatini
Eustachian tube lumen
Cartilage
Ext. auditory canal
Tympanic membrane
Malleus
Tensor tympani
Middle ear
Mastoid


INFANT EAR
Malleus
Incus
Stapes
Ext. auditory canal
Eustachian tube
Tympanic membrane
ADULT EAR

DIFFERENCES BETWEEN PAEDIATRIC AND ADULT EUSTACHIAN TUBE

	Infant	**Adult**
Length	13-18mm	31-38mm
Direction	More horizontal. Forms an angle of 10^0 with horizontal.	Slopes downwards forwards & medially. Forms angle of 45^0 with horizontal.
Angulation with isthmus	Absent	Present
Bony vs cartilagenous part	Bony part longer than cartilaginous part	Bony part-1/3 Cartilagenous -2/3
Tubal cartilage	Flaccid	Rigid
Density of cartilage at hinge	Less. Tubal closure less efficient	More. tubal closure effective

AETIO –PATHOLOGY:

Anterior and inferior part of the middle ear cavity and Eustachian tube is lined by the ciliated, pseudostratified columnar epithelium of the respiratory tract .These cells along with goblet cells, mucous glands secrete mucus.This mucous is removed by mucociliary transport into the nasopharynx via the eustachian tube. Secretory otitis media is primarily caused by factors resulting in an overproduction of mucus, an impaired clearance of mucus or both. Both viral and bacterial infection can lead to the increased production and viscosity of secretions from the middle ear mucosa. Infection also leads to inflammatory edema of the mucosa, which may obstruct the eustachian tube. Temporary paralysis of cilia by bacterial exotoxins further impedes the clearance of an effusion.

INFECTION:

Infective pathogens stimulate an immune response,with release of cytokines. Respiratory viruses may predispose to bacterial infection or may stimulate an immune response themselves. The release of inflammatory mediators upregulates the mucin genes expressed in the middle ear, leading to the secretion of mucin-rich fluid, recognised clinically as the middle ear effusion seen at myringotomy.

In a small study of middle ear effusions that produced “sterile” cultures, polymerase chain reaction (PCR) confirmed the presence of bacteria (like *Haemophilus influenzae, Streptococcus pneumoniae and Moraxella catarrhalis*) in apparently sterile middle ear fluid.

The incidence of pathogens was higher in younger children (less than two years) and those with recurrent upper respiratory infections and recurrent attacks of otitis media.

The following table shows a positive bacteriological culture of middle ear aspirate of OME fluid (more than two months duration) in children with a wide spectrum of organisms[21]

BACTERIOLOGY OF SECRETORY OTITIS MEDIA

Organisms cultured	**Percentage**
Streptococcus pneumoniae	8
Haemophilus influenzae	3
Branhamella catarrhalis	7
Streptococcus pyogenes	1
Other bacteria	15
Negative culture	66
Total	100

Confocal laser scanning microscopy and vital dye examination of middle ear tissue biopsy samples taken at the time of ventilation tube insertion have demonstrated biofilm colonies in 80% of ears .

Koch's postulates, using traditional methods of culturing bacteria as proof of infectious aetiology, is no longer a tenable approach to the confirmation of bacterial infection. More recent studies of culture-negative middle ear effusions found that PCR techniques could demonstrate bacterial mRNA as a marker for metabolically active organisms in culture-sterile middle ear effusions. In the context of a biofilm infection, the fulfilment of middle ear effusions, the inference is that bacteria, in a form that could not be previously demonstrated, are present even in "sterile" effusions, cultured by traditional techniques.

Much current evidence points to bacterial biofilm colonies as a major cause of middle ear inflammation and of persistence of middle ear effusion. Rosenfeld concluded that the benefits from antibiotic treatment in the secretory otitis media phase are so slight that, in the face of drug resistance from widespread low-dose use, the prescribing of antibiotics is a poor clinical policy. As evidence gathers that persistent secretory otitis media is likely to be largely due to a biofilm infection, there will be many implications for the future management of secretory otitis media. The use

of biofilm-resistant surfaces, probiotic treatment, chemical or mechanical disruption of the protective glycocalyx of the biofilm and ultimately, modulation of the biofilm phenotype are all modalities of treatment for secretory otitis media that have been proposed.

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EUSTACHIAN TUBE DYSFUNCTION:

Eustachian tube dysfunction can lead to poor aeration of middle ear cleft. Most common cause of damage to epithelium of Eustachian tube is secondary to viral upper respiratory tract infection, the other reasons can be secondary to an allergic reaction or pollutants (cigarette smoke), chronic nasopharyngeal infection in the adenoid or gastroesophageal reflux, disorder in palatine muscles.

ALLERGY:

Though it appears that allergic response is the primary casual factor in the development of secretory otitis media, the majority of children with OME do not have allergic symptoms of either upper or lower respiratory tract. In Population cohorts of children under the age of two years that have investigated atopy/allergy as a risk factor for persistence did not find atopy/allergy to be a significant factor.[22,23] Prospective studies of children covering all ages with secretory otitis

media that looked for risk factors for persistence did not find atopy/allergy to be a significant factor. [24,25,26,27,28].

CRANIOFACIAL ABNORMALITIES:

Secretory otitis media is invariably seen in every child with cleft palate due to deficient palatal muscles and resultant poor Eustachian tube function.Surgical correction of cleft palate does not influence the incidence of secretory otitis media. Downs and Turner syndrome are also more likely to have secretory otitis media, but bifid uvula do not appear to predispose it.

TUMORS OF NASOPHARYNX :

All benign and malignant tumors of nasopharynx can lead to Eustachian tube blockade and cause secretory otitis media .

GASTRO-OESOPHAGEAL REFLUX:

There is accumulating evidence that gastric acid may act as an inflammatory co-factor in the development of low grade inflammation of the Eustachian tube and middle ear mucosa. In a study of middle ear effusions at the time of myringotomy in 54 children aged 2–8 years, 83% of the effusions contained pepsin/pepsinogen at concentrations up to

1000-fold greater than those in serum.[30,31] Inflammation allows the establishment of a biofilm colony, resulting in persistence of the middle ear effusion. Inflammation secondary to acid reflux also promotes bacterial colonization.

SYMPTOMS AND SIGNS:

Secretory otitis media may be asymptomatic. Discomfort from the presence of the middle ear effusion does occur, but it is hard to measure, and clinical reports from older children suggest that it is not always an important symptom.Some may complain of hearing loss,less commonly,tinnitus.Sometimes the child has a behavioural disorder because of the hearing deficit and consequent inability to communicate adequately. In younger children, poor articulation, language delay and poor balance may be observed . In older children, nursery teachers or school teachers often draw attention to the hearing loss when inattention in a group setting is noted. Poor social interaction and disengagement from class activities that require good “signal in noise” hearing are frequently reported. Frequently, a hearing loss is noted at a routine hearing screening assessment, before which there was little parental concern about the child’s hearing.

DIAGNOSIS:

HISTORY:

History is not a reliable indicator of the current presence of secretory otitis media or the degree of hearing impairment. In a cohort study of 216 children followed from birth to 27 months of age at 3 months intervals the sensitivity of parental report of current OME and hearing improvement was poor. [32]

Clinical diagnosis following a full history is made by examination of the ears and age-appropriate audiological testing.

OTOSCOPY:

The tympanic membrane is best assessed with otomicroscopy, but in general clinical practice, a bright halogen otoscope provides clear illumination.Normal tympanic membrane appears pearly white in colour, concave and moves with pneumatic otoscopy. As effusion develops landmarks of tympanic membrane gets obscured,mobility gets decreased, air-fluid level appears. The foreshortening of handle of malleus that is often seen results from negative middle ear pressure drawing the long process of the malleus medially. Attic retraction or retraction of the posterior pars tensa of the tympanic membrane may also be visible. These findings do not necessarily indicate a more severe or persistent type of

secretory otitis media, and are not absolute indications for urgent surgical intervention.

Otoscopy alone is poorly predictive of the hearing loss associated with the presence of middle ear fluid, and while pneumatic otoscopy may aid diagnosis, audiometry is essential to assess the degree of hearing loss also serves as a marker for the wider impact and likelihood of resolution.

PNEUMATIC OTOSCOPY:

This can be done with either a hand held otoscope or with a siegle pneumatic speculum viewed with headlight illumination or microscope. Inability to achieve a seal with the available speculum can occur in upto 20% of children over 18 months.[34] Overall there is a relative improvement in the diagnosis with pneumatic otoscopy.

American clinical practice guidelines have strongly recommended the use of pneumatic otoscopy as the primary diagnostic method for OME.[35,36,37].

AUDIOLOGY:

In younger children, below the age of 2 years, visual reinforcement audiometry, using sound field testing or delivering the sound to each ear separately with “inserts”, and bone-conduction testing, produce reliable thresholds. Older children are conditioned using play audiometry in

sound field or with headphones and can also cooperate with speech testing using the toy identification test.

TYMPANOMETRY:

Tympanometry alone is a useful screening tool in the investigation of secretory otitis media. It is easy to use, provides reproducible results, is inexpensive, and is widely tolerated by patients—even young children. By measuring the compliance of the middle ear transformer mechanism, it provides an objective assessment of the status of the middle ear. Tympanometry produces a peak (ie, maximal compliance) when the pressure in the external ear canal equals that of the middle ear. By varying the pressure in the external ear, the tympanometer is able to provide information on the status of the middle ear. If there is an effusion in the middle ear, then compliance does not vary with changes in canal pressure, and a flat (Type B) tympanogram is produced. If the air in the middle ear is at or near atmospheric pressure, then a normal (Type A) tympanogram is produced. Negative middle ear pressure results in a Type C tympanogram, with the compliance peak being at less -99 da pa.

Tympanograms can be classified in multiple ways, the simplest being peaked/non peaked, usually with the additional subclassification of peaked tympanograms depending on the pressure at which the peak is recorded.

TYPE	DESCRIPTION
PEAKED	
A	between +200 and -99 dapa
C_1	between -100 and -199 dapa
C_2	between -200 and -399 dapa
NON PEAKED	
B	no observable peak between +200 and -600 dapa

TREATMENT:

Any treatment regimen should take into account not only the severity of disease, but also the persistence of constant or intermittent disease, whether the condition is symptomatic.Such symptoms may concern hearing loss or its secondary effect on speech, language, cognition and behaviour.

Treatment should aim to relieve the symptoms caused by the middle ear condition, to resolve the underlying pathophysiological changes and to prevent recurrence and development of sequelae.

MEDICAL MANAGEMENT:

Medical treatment will be of immense value if it speeds the resolution of an episode of secretory otitis media. Most trials follow up children for one or two weeks after therapy. If at this point the therapy is ineffective, there is no reason for further follow up as it is unlikely to be of benefit thereafter. However, if it is effective after one or two weeks, then follow up for the recommended watchful waiting period of 12 weeks is necessary to see if it is of benefit in the longer term and might be used to reduce the proportion of children being considered for surgery.

ANTIBIOTICS:

Secretory otitis media, like Acute otitis media, is a bacterial disease, and the Middle ear effusion is known to contain viable, pathogenic bacteria.[38] Antimicrobial therapy is a logical choice, and the efficacy of this therapy has been determined in several reports. In an uncontrolled study using the combination of erythromycin ethylsuccinate and sulfisoxazole (Pediazole), Gates and others [39] found that 45% of cases cleared by 1 month and 60% cleared by 2 months In placebo-controlled trials by Healy [40] using trimethoprim-sulfisoxazole, by Mandel and others[41] using amoxicillin, and by Thomsen and others.[42] using amoxicillin-clavulanate, the clear rates in the treated cases in each study

were significantly greater than in the control groups. However, the magnitude of the difference was not great.[43,44] The AHCPR [45] recommended a course of antibiotics (optional for children with asymptomatic OME) followed by at least a 1-month observation period. If signs of improvement are noted by otoscopy or tympanometry, additional observation may be warranted. Surgical treatment may be considered if the effusion persists and is associated with hearing loss.

ANTIHISTAMINES AND NASAL DECONGESTANTS:

To determine whether systemic medical therapy is of value in patients with secretory otitis media, Bluestone, Cantekin, and Beery [48] randomly assigned 553 infants and children with secretory otitis media to receive either a decongestant-antihistamine or placebo. The clearance of effusion did not differ between the two groups. As a consequence of this study, the routine use of decongestants in children with secretory otitis media has been abandoned.

CORTICOSTEROIDS:

The benefit of a short course of prednisolone (1 mg/kg maximum dose) to aid the clearance of persistent secretory otitis media appears, at best, to be slight and temporary.[49,50] There is no evidence to suggest that oral steroids are effective for longer term than in the short term (two

weeks) even combined with antibiotics.[51,52,53,54] Also steroid treatment has the potential complication of adrenal suppression and the development of varicella.Systemic steroids cannot be recommended at present for childhood secretory otitis media.

ADENOIDECTOMY

Adenoidectomy is being increasingly used for the treatment of secretory otitis media because recent studies have confirmed its effectiveness.[55,56,57] Adenoidectomy was once the principal surgical treatment for OM. With the widespread use of tympanostomy tubes, however, adenoidectomy was used far less in certain countries. Probable reasons for this difference were the following: adenoidectomy takes longer time than tympanostomy tube insertion, adenoidectomy carries a risk of hemorrhage and other complications such as hypernasality, the mechanism of its effect on the middle ear is not well understood, and early studies did not demonstrate effectiveness.[58,59,60]

The rationale for removal of the adenoid in children with otitis media has been enlargement causing nasal obstruction and mouth breathing. Removal of a large adenoid that occludes the nasopharynx will open the airway and relieve the nasopharyngeal overpressure that occurs with closed-nose swallowing.[61] Removal should also lessen eustachian

tube reflux. Although the proportion of children with otitis media caused by reflux is unknown, this rationale for adenoidectomy is logical. The association of enlargement with abnormality has been called into question, however, because adenoid and tonsil enlargement result from clonal expansion of immunocompetent cells.[62] This suggests that the large adenoid may be more immunocompetent than the small adenoid because chronic infection is associated with cellular depletion. Brandtzaeg and Berdal[63] demonstrated a general decline in immunocytes in the tonsils of patients with recurrent tonsillitis. Thus, current knowledge suggests that adenoid enlargement, a common phenomenon in 4- to 7-year-old children,[64] does not necessarily indicate abnormality. Basing the rationale for adenoidectomy in children with otitis media on size alone, therefore, has little scientific basis. Further, clinical evidence from three separate studies [65,66,67] indicates that the effect of adenoidectomy is independent of adenoid size. Removal of a large adenoid is a clinically attractive option because it offers symptomatic relief to a compromised airway. The removal of a small, chronically infected adenoid offers no obvious benefit except in controlling otitis media.

The other classic rationale for adenoidectomy is improvement in eustachian tube function. Honjo[68] showed improvement in equilibration

of positive middle ear pressure after adenoidectomy but no change in the ability to equilibrate negative pressure and no change in the static opening pressure of the tube. Obstruction of the eustachian tube, either anatomic or functional, is a logical rationale for the procedure. Bluestone and others [69] showed that obstruction of the eustachian tube is unusual in most children with secretory otitis media. The obstruction is functional in most cases. Eustachian tube function tests are not available except in research centers, and thus eustachian tube function is not routinely tested preoperatively. In children with hypercompliant eustachian tubes, adenoidectomy may increase reflux, particularly if the tissue in Rosenmüller's fossa is removed.[70]The third and most current rationale is removal of the chronically infected adenoid to eliminate a nasopharyngeal source of infection.[71]Adenoidectomy should result in a smooth lining of the nasopharynx, which should decrease bacterial colonization of the nasopharynx and, indirectly, the middle ear. Further research is needed to develop the methodology to identify subgroups of children who might benefit the most from adenoidectomy for control of infection.

Three studies[72 ,73,74]of the efficacy of adenoidectomy have demonstrated consistent results. Other studies, which showed a lack of effect of adenoidectomy,[75,76,77] lacked statistical power to detect differences among

the treatment groups that may have been clinically significant. Other older studies of adenoidectomy[78,79,80] are not reviewed here because of severe flaws in experimental design.

MYRINGOTOMY:

Myringotomy with aspiration has not been shown to be effective in restoring the hearing levels in children with secretory otitis media.

Evacuation of the middle ear effusion by myringotomy and suction aspiration has been studied as one type of therapy. However, the results from this simple procedure have been disappointing.[81] Most investigators and clinicians agree that if a child is to receive an anesthetic for such treatment, then tympanostomy tubes should be inserted or an adenoidectomy performed (or both) because the cost-benefit ratio for myringotomy and aspiration is too low to justify myringotomy as an independent procedure.

TYMPANOSTOMY TUBES

Modern tympanostomy tubes were introduced by Armstrong[82] in 1954 and have become the therapeutic gold standard and the most widely used treatment option for secretory otitis media. Improved hearing and a decreased rate of recurrent acute otitis media are predictable benefits of their use. The complications of tympanostomy tubes (purulent otorrhea,

permanent perforation, focal drum atrophy, and recurrent effusion), while minor, are a cause of concern. The goal of using tympanostomy tubes is prolonged ventilation of the tympanum. Removal of the middle ear effusion and restoration of an aerated tympanum results in prompt return of hearing to preinfection levels in the vast majority of patients. Experimental evidence suggests that the mucosal hyperplasia of the tympanum will revert to a more normal condition with aeration.[83]Once the tubes are extruded, however, the clinical benefit appears to end.[84]

Many early investigators compared the effects of tympanostomy tubes in one ear and myringotomy in the opposite ear in children undergoing adenoidectomy.[85,86,87] The conclusions of these studies varied because the manifestations of otitis media may vary in the same child. Using a different design in which patients were compared, Gates and others[88] found a significantly better outcome in terms of hearing, less time with middle ear effusion, longer time to recurrence, and fewer repeat operations in the children with tympanostomy tubes as compared with those undergoing myringotomy and aspiration. Similar conclusions were reached by Mandel, Bluestone, and Paradise.[89] Paradise[90] and Paradise and others[91] argued that tympanostomy tubes should be used as the initial procedure of choice for patients with persistent otitis media with effusion, because tympanostomy tube placement is less invasive and less expensive

than adenoidectomy. They reserve adenoidectomy for cases of recurrent secretory otitis media.

SEQUELAE OF SECRETORY OTITIS MEDIA :

Chronic otitis media with effusion can lead to middle ear atelectasis, retraction pocket.This ultimately leads on to cholesteatoma formation**.** Tympanostomy tubes which are used in the treatment of otitis media with effusion can lead onto tympanosclerosis,permanent perforation, chronic otorrhea.

TOS CLASSIFICATION OF ATTIC RETRACTION POCKETS:

Stage I	Mild retraction, with air still present between the pocket and malleus neck.
Stage II	Pocket touches malleus neck with or without erosion of neck.
Stage III	Pocket begins to expand: limited erosion of outer attic Wall.
Stage IV	More severe erosion ofouter attic wall ,pocket attached to malleus head.

SADE CLASSIFICATION OF TYMPANIC MEMBRANE ATELECTASIS :

Grade	Description
Grade 1	The “retracted drum”
Grade 2	The drum is touching or adhering to the incus or stapes.
Grade 3	The drum is touching the promontory.
Grade	The drum adheres to the promontory.

MATERIALS AND METHODS

This study was done in the Department of ENT, Coimbatore Medical College, Coimbatore. It consists of 50 patients and their age ranges from 4years to 50 years.

INCLUSION CRITERIA:

1. Patients with complaints of hard of hearing/discomfort or blocking sensation of the ear for more than 3 months.
2. Otoscopic evidence of secretory otitis media
3. An impendance audiometry with type ‘B’ or ‘C’ curve.

Patients with combination of two or more of these criteria were included in the study.

EXCLUSION CRITERIA:

1. Patients with acute ear pain,ear discharge.
2. Patients deaf since childhood or with family history of hard of hearing.
3. Patients with cleft palate,beningn and malignant tumors of nasopharynx.

A detailed history was recorded for all patients. Past surgical history was also noted. All patients were examined with a pneumatic otoscope and the findings were recorded in 3 formats as

1. Normal tympanic membrane
2. A thin semitransparent tympanic membrane with air bubble or fluid level.
3. A dull or yellowish/opaque,retracted/bulging lustreless tympanic membrane with distorted or absent cone cone of light

Other clinical examination like tuning fork tests, nasal, oral cavity examination were performed. Patients with allergy, chronic sinusitis, chronic adenotonsilitis were segregated.

All patients were subjected to pure tone audiometry and graphical recording of their hearing threshold were made and the pure tone average in both ears were recorded. Tympanometry was done in all patients.

Both tympanometry and acoustic reflex testing were done. Radiological investigations in the form of X-ray PNS (occipito mental view) were done in suspected cases of chronic sinusitis and assessment were made regarding pharyngeal end of Eustachian tube orifice and adenoids by diagnostic nasal endoscopy.

Examination under microscope was done to confirm the otoscopic findings. The predisposing factors for otitis media with effusion if present were noted.

One group of patients (25 Nos) were given medical treatment for 6 weeks which included

1. Antibiotics,anti inflamatory drugs,antihistamines,nasal decongestants.
2. They were instructed to perform valsalva manoeuvre ,3-5 times a day.

Improvement in medical treatment is considered if

1. Patients have symptomatic relief.
2. Otoscopically tympanic membrane become normal
3. Pure tone audiometry showed good improvement in hearing air bone gap shows <10 dB

If patients satisfy these criteria, improvement was further confirmed by tympanometry.

Other group of patients (25 Nos) were subjected to Surgical management which includes :

1. Myringotomy and grommet insertion
2. Adenotonsillectomy with myringotomy and grommet insertion(Shephards)

These patients were followed regularly.Success of the procedure was assessed by following means

1. Symptomatic improvement
2. Otoscopic evidence of improvement.
3. Air bone gap <10dB in postoperative audiogram done after 3 weeks.

RESULTS AND ANALYSIS

This study is the prospective analysis of the incidence, predisposing factors, clinical profile and the treatment outcome of medical and surgical management conducted in the Department of ENT, Coimbatore Medical College Hospital, Coimbatore.

The demographic profile shows the most common age group affected was between 5 to 15 years. Of the 50 patients studied 58% (29 patients) were male and 42% (21 patients) were female. In the various age groups there was no significant difference between male and female patients.

The most common symptom was hard of hearing which was seen in 68% of patients followed by ear fullness (54%), otalgia (48%), nasal symptoms (26%). On pneumatic otoscopic examination, most common sign was impaired tympanic membrane mobility which accounted for 92% of patients, followed by retracted tympanic membrane (60%), fluid level (44%).

Eustachian tube function was assessed by diagnostic nasal endoscopy and pneumatic otoscopy. Adenoid hypertrophy was the most common predisposing factor found in 66% of patient, followed by allergy (12%), GERD (8%).

Though cleft palate is a risk factor for secretary otitis media, we did not encounter any patient with cleft palate.

Majority of the patients had hearing loss in the range of 20-40 db (69%). About 77% of patients had B curve, 23% patients had 'C' curve.

Our patients were randomized into medical treatment arm and surgical treatment arm, and the results were analyzed in terms of symptomatic relief, pure tone audiogram results and pneumatic otoscopy. Out of 25 patients, who were taken up for medical treatment 60% of patients showed a significant reduction in the air bone gap with air bone gap less than 10 db as compared to the pretreatment values. 52% of the patients had their tympanic membrane returned to normal appearance. Only 20% of the patients had symptomatic relief.

Considering the patients with 2 of the 3 factors (symptomatic relief, tympanic membrane returned to normal appearance, air bone gap less than 10 db) as successful outcome of medical treatment, only 40% (10 patients) had successful outcome. Patients in surgical treatment arm (25 No's) along with patients (15 No's) who were declared as failure of medical management, a total of 40 patients were subjected to surgical management.

Out of the 40 Patients who underwent surgical treatment 70% showed a significant improvement when the above criteria for successful outcome was taken. Of the 15 patients who were taken up for surgical treatment because of failure of medical treatment, 4 patients did not show significant improvement even after surgical treatment.

TABLES & CHARTS

Table 1

Age Distribution

Age in Years	No.of Patients
05 - 10	20
11 - 15	10
16 - 20	6
21 - 25	2
26 - 30	3
31 - 35	1
36- 40	4
41 - 45	1
46 - 50	2
51 - 55	1
Total	**50**

Chart 1

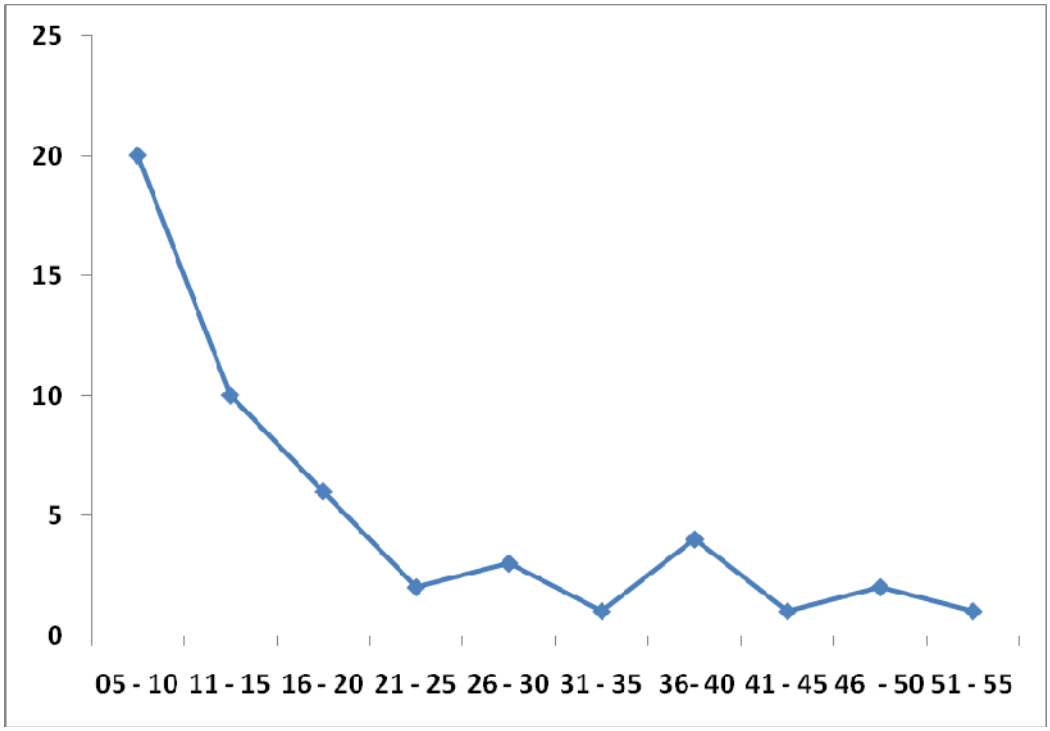

Table 2

Sex Distribution

Sex	No.of Patients
Male	29
Female	21

Chart 2

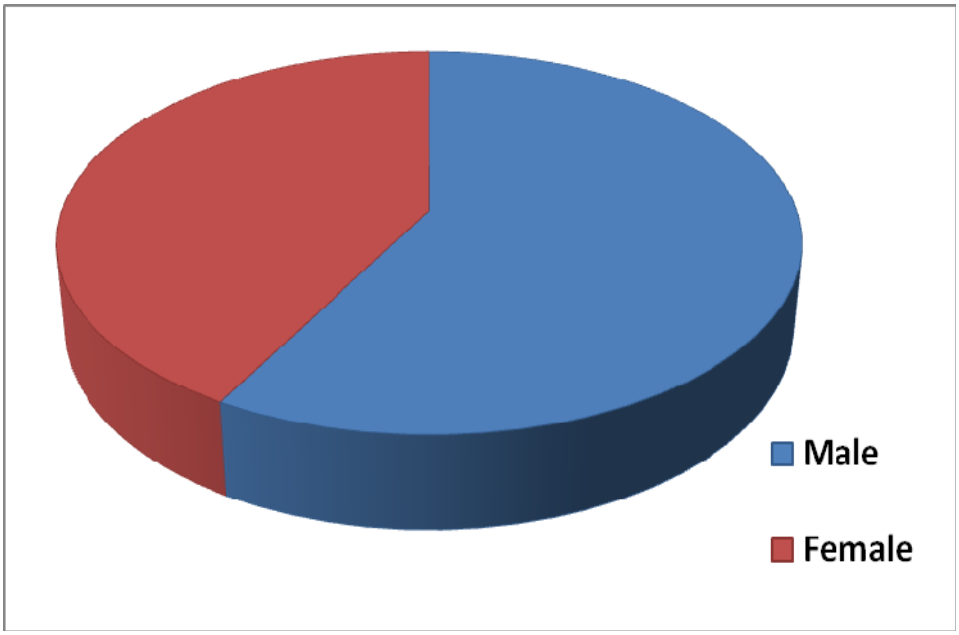

Table 3

Age Group /Sex Distribution

Age	Male	Female
1 – 10	10	10
11 – 20	9	7
21 – 30	4	1
31 – 40	3	2
41 – 50	1	2
51 – 60	1	0

Chart 3

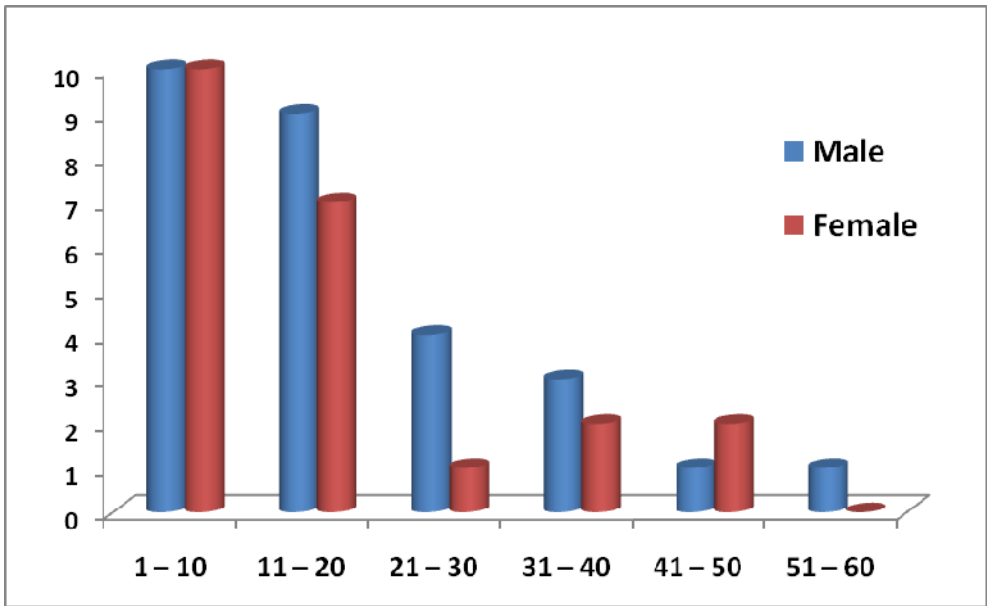

Table 4

Symptomology

Symptoms	No.of Patients
Hard of Hearing	34
Otalgia	24
Ear Fullness	27
Nasal Symptoms	13

Chart 4

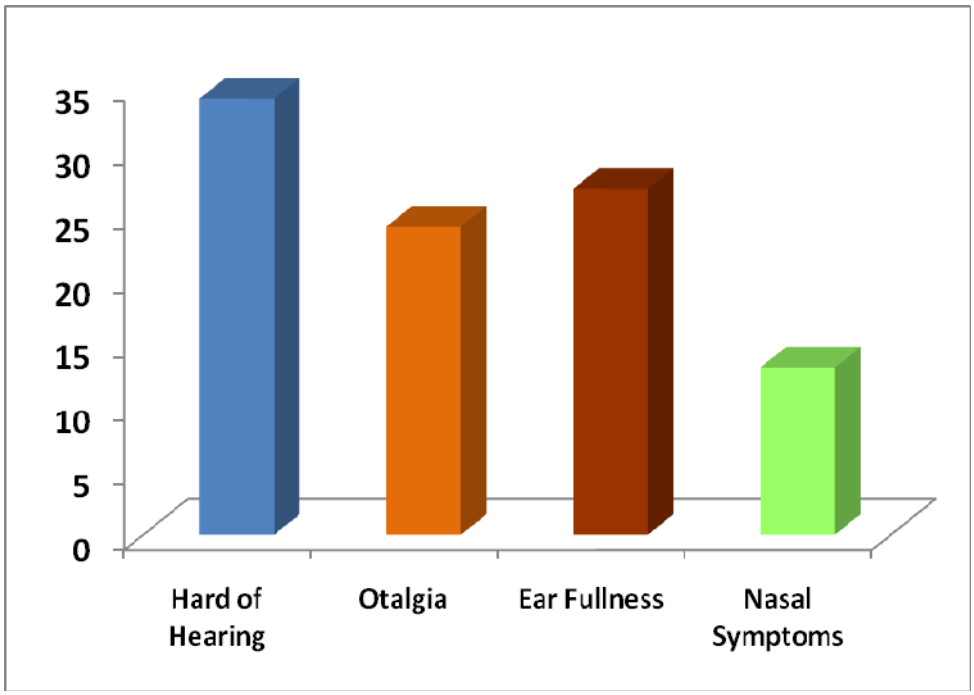

Table 5

Signs

Sign	No.of Patients
Fluid Level	22
Immobility	46
T.M. Retraction	30

Chart 5

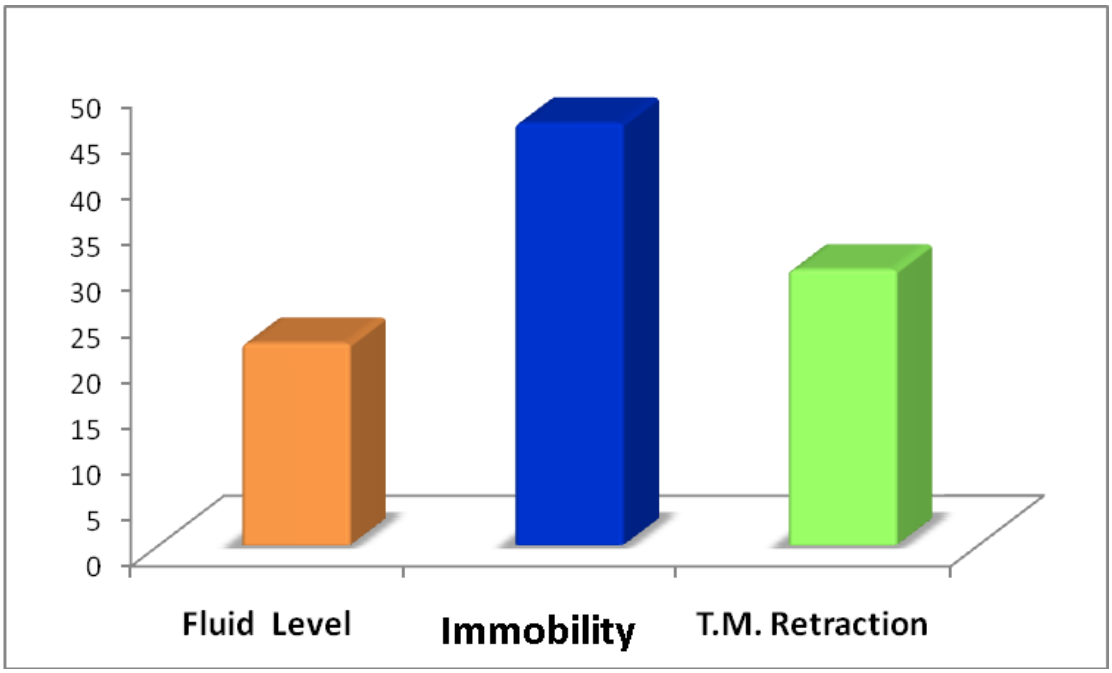

Table 6

Pre Disposing Factors

Predisposing Factors	No.of Patients
ET - Dysfunction	33
Allergy	12
Cleft Palate	0
GERD	8

Chart 6

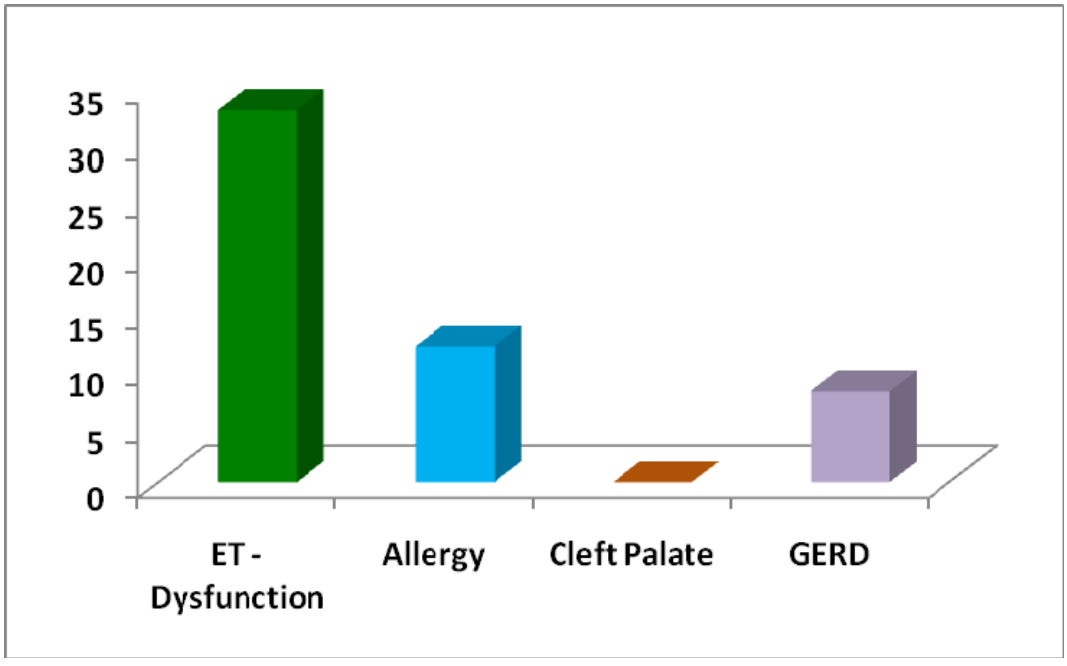

Table 7

Medical management-Results

Results of Medical Treatment	No.of Patients
Symptomatic Relief	7
TM Normal	13
Air bone gap < 10 db	15

Chart 7

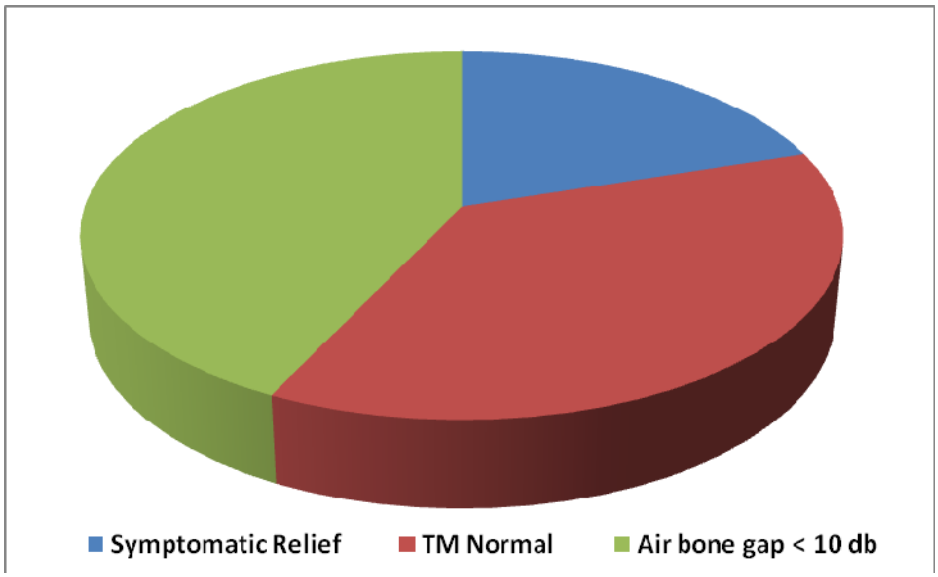

Table 8

Surgical Management-Results

Results of Surgical Treatment	No.of Patients
Symptomatic Relief	13
TM Normal	25
Air bone gap < 10 db	27

Chart 8

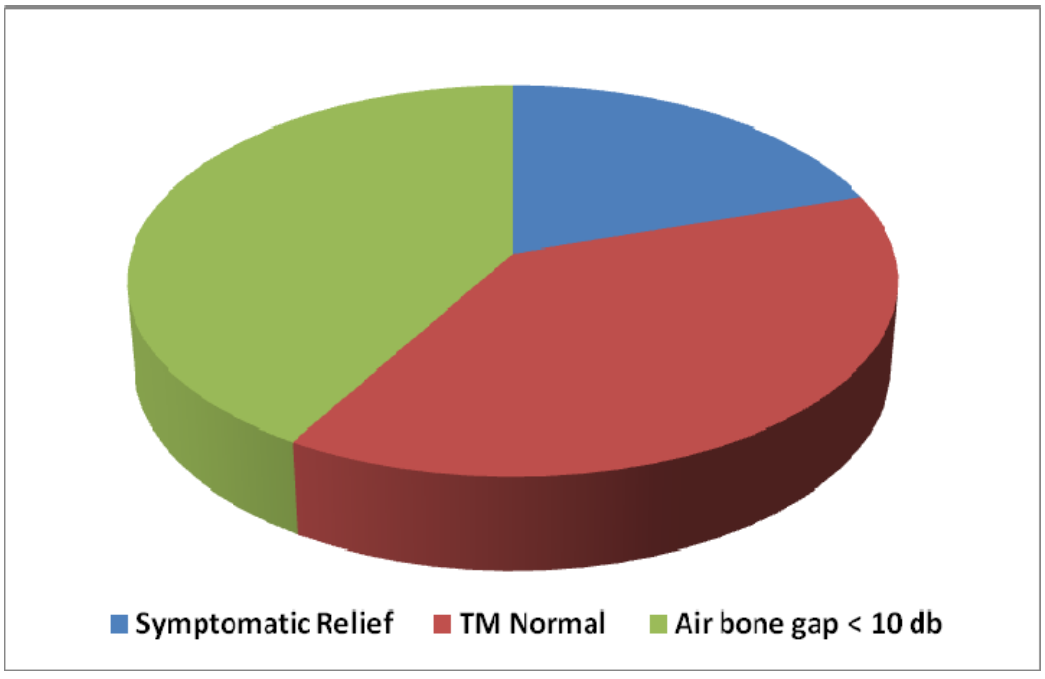

Table 9

PTA Evaluation

Hearing Loss in db	No.of Patients	
	R	L
20 – 30	13	14
31 – 40	22	20
41 – 50	10	7
51 – 60	3	0
61 - 70	0	0

Table 10

Tympanometry-Results

Tympanometry	No.of Patients	
	R	L
A	0	0
Ad	0	0
As	0	0
B	36	41
C	14	9

DISCUSSION

Zielhius et al[3] reviewed about 23 studies which used tympanometry as one of the diagnostic tool to give age specific prevalence rates and found that the prevalence is bimodal with first peak, at 2 years. When child attends a play group or nursery school, second peak, at 5 years of age when the child attends a primary school. But in our study there was no bimodal prevalence. About 60% of the patient were in the age group 5-15 years.

According to Tos et al,[4] Rovers et al,[5] secretory otitis media shows a increased prevalence in temperate climate when compared to summer, probably due to increased incidence of upper respiratory tract infection. Engels et al,[14] Rovers et al,[15] has suggested that there is less more risk in boys when compared to girls. In our study also incidence was more in boys (58%)

Bluestone et al has proposed that there might be a casual relationship between parental smoking and both acute and chronic middle ear disease in children. But in some study by Engel, has found that when the other factors have no effect of parental smoking is detected.

Eustachian tube dysfunction can lead to poor aeration of middle ear cleft. Most commonly it is secondary to Upper Viral Respiratory infection, Allergy, Adenoid infection, GERD or disorder in palatine

Muscles. In our study, ET dysfunction was the major predisposing factor seen in 66% of patients followed by allergy (12%), GERD (8%).

American academy of paediatrics has strongly recommended the use of pneumatic otoscopy as the primary diagnostic method. Tympanometry alone is a useful screening tool in the investigation secretory otitis media.[36]

Wafters GW, Jones JE in published in clinical otolaryngology about the predictive values of tympanometry in the diagnosis secretory otitis media. A type B tympanogram has a high sensitivity (0.91) in predicting middle ear effusion with good specificity (0.3) type 'C' increases the sensitivity of predicting dry middle ear to 0.79. In our study, type B curve was found in 77% and type 'C' curve was found in 23%.

Gates and other[39] found that in 45% of cases treated with antibiotics erythromycin ethylsuccinate and sulfisoxale), effusion cleared by one month and 60% cleared in two months. Mandle et al [42] in their study found that with amoxicillin, the clearance of effusion was significantly greater in the control groups. Blue stone et al [48] in their study found that the clearance of effusion did not differ between the groups who received a decongestants – antihistamine and placebo.

Lambert et al, Rovers proposed that there is a short term benefit with Prednisolone (1mg/kg) clearance of the effusion is sling and termporary

Basing the rationale for adenoidectoimy in children with Otitis media on size alone, has little scientific basis. But Gates et al, paradise et al, have demonstrated the effectiveness of adenoidectoimy in the management of secretary otitis media. Further clinical evidence from the above studies indicates the effect of adenoidectomy is independent of adenoid size. The other classic rationale is improvement in Eustachian tube function. Honjo [68] showed improvement in equilibration of positive middle ear pressure after adenoidectomy but no change in the ability to equilibrate negative pressure and no change in the static opening pressure of the tube. Obstruction of the Eustachian tube either anatomic or functional is a logical rationale for the procedure.

The goal of using tympanostomy tubes is prolonged ventilation of the tympanum. Removal of the MEE and restoration of an aerated tympanum results in prompt return of hearing the pre infection levels in the vast majority of petients. Experimental evidence suggests that mucosal hyperplasia of the tympanum will revert to a more normal condition with aeration.[83] Once the tubes are extruded however, the clinical benefit appears to end.[84]

Gates and others [88] found a significantly better outcome in terms of hearing, decreased duration of middle ear effusion, longer time to recurrence, and fewer repeat operations in the children with tympanostomy tubes as compared with those undergoing myringotomy and aspiration. Similar conclusions were reached by Madel, Bluestone, and Paradise.[89] Paradise[90] and paradise and others[91] argued that tympanostomy tubes should be used as the initial procedure of choice for patients with persistent OME, because tympanostomy tube placement is less involved and less expensive than adenoidectomy. They reserve adenoidectomy for cases with recurrent secretary otitis media.

SUMMARY

In our Study, the predominant age group was found to be between 5 -15 years . There was no significant difference in the incidence among males(58 %) and females (42%). The most common symptom was hard of hearing (68%) followed by otalgia (48%), nasal symptoms (26%). Most common sign was impaired Mobility(92%) followed by retracted tympanic membrane(60%) and fluid level/air bubbles(44%). Among the predisposing factors ET - dysfunction was found in 66% of patients. About 69% of the patients had hearing loss in the range of 20-40 db while 77% of patients had 'B'curve, 23% of the patients had C' curve. Medical treatment showed a successful outcome in 40% of patients. The remaining 60% patients who did not show improvement were subject to surgical treatment out of the 40 patients who underwent s surgical treatment, 70% had successful outcome.

CONCLUSION

From our study it is evident that secretory otitis media is a treatable cause of conductive hearing loss in children. In children, Eustachian tube dysfunction was secondary to functional or mechanical obstruction was the common precipitating factor for secretory otitis media. GERD was found to be associated with almost all adult patients. It has to be studied whether control of acid reflux can have any effect in these patients. From our study it was evident that medical management helps in the control of acute episodes of secretory otitis media which is associated with frequent relapse and recurrence.

Surgical management in the form of adenoidectomy / myringotomy with grommet insertion has better long term outcome in terms of hearing impairment and disease relapse and recurrence. In our study we used Shepards tympanostomy tubes which was associated with extrusion rate of 10% at 3 months follow up period. Further studies are needed to compare the efficacy of various tympanostomy tubes on disease control and various complications. A long term follow up is needed to assess the disease relapse and recurrence, also to study the various sequlae of secretory otitis media like adhesive otitis media and cholesteatoma.

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PROFORMA

NAME: AGE: SEX:

COMPLAINTS: Hard of hearing / blocking sensation of ears.

Tinnitus.

Otalgia.

Associated symptoms-nasal obstruction

-nasal discharge.

HISTORY OF PRESENTING ILLNESS:

1.HARD OF HEARING :

- Side
- Duration
- Onset-sudden/insidious/progressive
- Ototoxic drugs
- Fluctuant deafness
- Paracusis willisii
- Autophony.

2.TINNITUS:

- Side
- Duration
- Type
- Onset- sudden insidious/progressive
- Character- intermittent/pulsatile/clicking
- Aggravating /relieving factors

3.OTALGIA:

- Side
- Duration
- Onset-sudden/gradual/progressive
- Aggrevating factors/relieving factors

4.VERTIGO:

- Duration
- Onset- sudden /gradual

5.NASAL OBSTRUCTION:

- Side
- Duration
- Onset-sudden/gradual/progressive
- Associated with mouth breathing
- Continous/intermittent

6.NASAL DISCHARGE:

- Side
- Duration
- Onset
- Type

HISTORY OF PREVIOUS ILLNESS:

- Allergy
- Asthma
- ASOM
- Hypertension
- Diabetes
- Pulmonary TB
- Trauma
- Irradiation
- Previous Surgery
- Travel by flight
- Hill travel

FAMILY HISTORY: Smoking, Allergy.

SOCIO ECONOMIC HISTORY:

GENERAL EXAMINATION:

Build, Anaemia, Jaundice, Cyanosis,

Generalised Lymphadenopathy.

Cardiovascular system:

Respiratory system:

Central nervous system:

EXAMINATION OF EAR: RIGHT LEFT

Pinna

Preauricular region

Postauricular region

External auditory canal

Tympanic membrane

Pars tensa

Pars flaccida

Handle of malleus

Lustre

Cone of light

Retraction

Movement

TUNING FORK TESTS :

Rinne test:

Weber test:

Absolute bone conduction tests:

EXAMINATION OF NOSE:

Anterior rhinoscopy:

Posterior rhinoscopy:

EXAMINATION OF THROAT:

- Oral hygiene
- Dental formula
- Tongue
- Tonsil
- Tonsillar pillar
- Palate(hard/soft)
- Posterior pharyngeal wall.

Indirect laryngoscopy:

INVESTIGATIONS:

Pure tone audiometry

Impedance audiometry

X-ray PNS

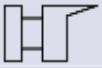
X-ray Nasopharynx lateral view for adenoids

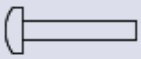
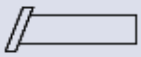
Diagnostic nasal endoscopy

CT- SCAN PNS

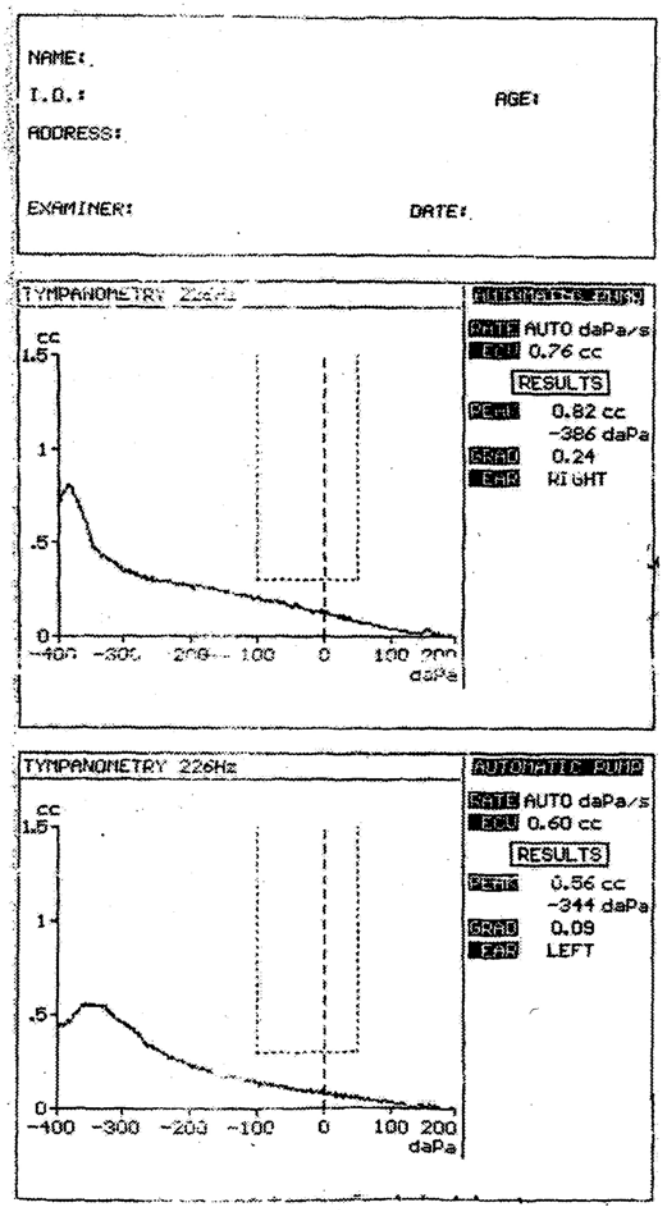
FOLLOW –UP: Otoscopy / Audiogram / Tympanometry.

MASTER CHART

	T-Tube (12mm)	Silicone	1.10 x 9.00 x 12.00 mm
	T-Tube (9 mm)	Silicone	1.10 x 7.50 x 9.00 mm
	T-Tube (6 mm)	Silicone	1.10 x 7.50 x 6.00 mm
	Paparella Tube I	Silicone	1.20 x 2.15 x 1.19 mm
	Paparella Tube II	Silicone	1.50 x 4.50 x 1.10 mm
	Shepard Grommets with tab	Silicone	1.10 x 2.30 x 1.50 mm
	Shepard Grommets with wire	Fluoroplastic	1.10 x 2.30 x 1.50 mm
	Donaldson	Silicone	1.14 x 2.30 x 0.80 mm
	Donaldson with tab	Silicone	1.14 x 2.30 x 0.80 mm
	Shah without wire	Fluoroplastic	1.10 x 3.15 x 1.55 mm
	Shah with wire	Fluoroplastic	1.10 x 3.15 x 1.55 mm
	Straight Tube	Fluoroplastic	0.90 x 2.20 x 7.00 mm

	Straight Tube	Fluoroplastic	1.14 x 2.40 x 7.00 mm
	Armstrong	Fluoroplastic	1.10 x 2.60 x 3.80 mm
	Armstrong (plain end)	Silicone	1.15 x 2.70 x 10.00 mm
	Shepard with tail	Silicone	1.20 x 2.40 x 1.50 mm
	Shepard Grommets without wire	Fluoroplastic	1.20 x 2.40 x 1.50 mm
	Donaldson	Fluoroplastic	1.10 x 2.30 x 0.90 mm
	Reuter Bobbin	Fluoroplastic	1.00 x 2.50 x 0.90 mm
	Collar Button	Fluoroplastic	1.25 x 2.90 x 1.30 mm

TYMPANOGRAM – C



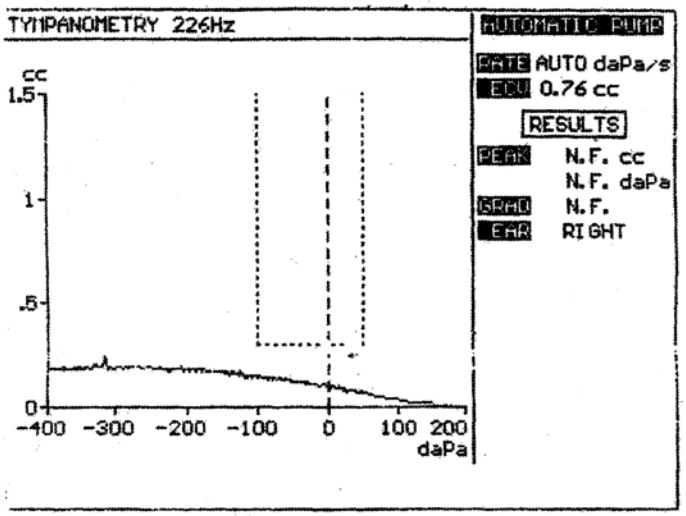
TYMPANOGRAM – B

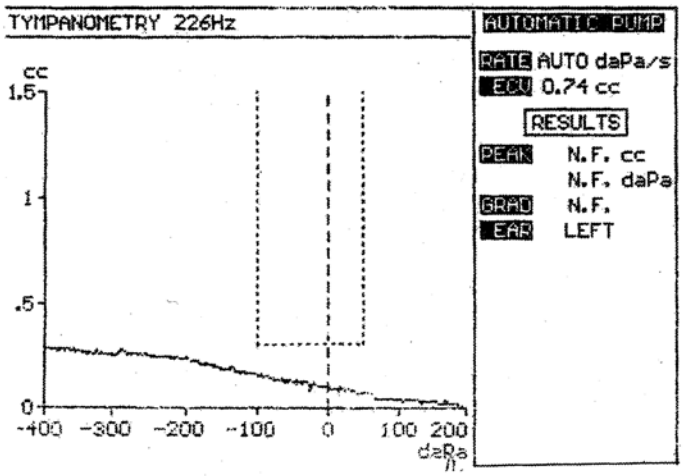
NAME:_

I.D.: AGE:_

ADDRESS:

EXAMINER: DATE:





S. NO	NAME	AGE	SEX	HISTORY				OTOSCOPIC FINDINGS					DIAGNOSTIC NASAL ENDOSCOPY				X-RAY PNS	X-RAY SKULL LAT VIEW FOR ADENOIDS	PTA -CONDN HEARING LOSS IN DB		TYMPANOMETRY - CURVE										PREDISPOSINGFACTORS				MEDICAL TREATMENT			SURGICAL TREATMENT DONE			SURGICAL TREATMENT RESULTS		
				HOH	OTALGIA	EAR FULLNESS	NASAL SYMPTOMS	COLOUR OF TM	RETRACTION	CONE OF LIGHT	AIR BUBBLES/ FLUID LEVEL	MOBI LITY	DNS	SINUSITIS	ALLERGIC RHINITS	ADENOID HYPERTROPY	SINUSITIS	ADENOID HYPERTROPHY	RIGHT	LEFT	RIGHT					LEFT					ET - DYSFUNCTION	ALLERGY	CLEFT PALATE	GERD	SYMPTAMATIC RELEIF	TM - NORMAL	AB-GAP <10DB	MYRIN GOTOMY	MYRINGOTOMY WITH GROMMET	ADENOTON SILECTOMY WITH GROMMET	SYMPTAMATIC RELEIF	TM - NORMAL	AB-GAP <10DB
																					A	B	C	AS	AD	A	B	C	AS	AD															
1	SUDHARSAN	13	MALE	+	-	-	-	DULL	+	-	-	-	-	-	-	+	-	+	48	42	-	+	-	-	-	-	+	-	-	-	+	-	-	-	-	-	+	+	+	+
2	ARUNACHALAM	9	MALE	+	-	-	-	DULL	+	-	+	-	-	-	-	+	-	+	18	38	-	+	-	-	-	-	+	-	-	-	+	-	-	-	-	-	+	-	+	+
3	NITHYA	14	FEMALE	+	-	-	-	DULL	+	-	-	-	-	-	-	+	-	+	18	18	-	+	-	-	-	-	+	-	-	-	+	-	-	-	-	-	+	+	-	+
4	NANDAKUMAR	20	MALE	+	+	+	-	AMBER	+	-	-	-	-	-	+	-	-	-	38	48	-	-	+	-	-	-	+	-	-	-	-	+	-	-	+	-	-	-	-	-
5	INDIRANI	48	FEMALE	-	+	+	-	AMBER		-	-	-	-	-	+	-	-	-	34	40	-	+	-	-	-	-	+	-	-	-	+	+	-	+	+	+	-	-	-	-	-
6	SRIMATHI	10	FEMALE	+	+	+	-	AMBER	+	-	-	-	-	-	+	-	-	-	46	40	-	-	+	-	-	-	-	+	-	-	+	+	-	+	+	-	-	-	-	-	-
7	VISWA	6	MALE	+	-	+	+	DULL	+	-	+	+	-	-	-	+	-	+	50	20	-	-	+	-	-	-	+	-	-	-	+	-	-	-	-	-	-	-	+	+	-
8	KEERTHANA	8	FEMALE	+	+	+	-	DULL	-	+	+	-	-	-	-	+	-	+	27	10	-	+	-	-	-	-	+	-	-	-	-	-	-	+	-	-	-	-	+	-	+
9	NIVETHA	12	FEMALE	+	-	+	+	DULL	+	-	-	-	-	-	-	+	-	+	53	15	-	+	-	-	-	-	+	-	-	-	-	-	-	+	-	-	-	-	+	-	+
10	DEIVANAI	30	FEMALE	+	+	-	-	DULL	+	-	-	-	-	-	+	-	-	-	45	18	-	+	-	-	-	-	+	-	-	-	+	-	-	-	-	-	-	+	-	+	+
11	BEULA	10	FEMALE	-	+	-	+	AMBER	-	-	+	-	-	-	-	+	-	+	38	28	-	+	-	-	-	-	-	-	+	-	-	-	-	-	-	+	-	-	+	-	+	+
12	NANDHINI	10	FEMALE	+	-	-	+	AMBER	-	-	+	-	-	-	-	+	-	-	38	46	-	+	-	-	-	-	+	-	-	-	+	-	-	-	-	+	+	-	-	-	-
13	SUBASH	11	MALE	+	+	-	-	DULL	+	-	-	-	-	-	-	+	-	+	23	23	-	-	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	+	+
14	KISHORE	9	MALE	+	+	+	-	DULL	+	-	-	-	-	-	-	+	-	+	38	17	-	+	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	+	-	+	+
15	MALINI	16	FEMALE	+	-	-	-	DULL	+	-	-	-	-	-	-	+	-	+	32	36	-	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	-	+	+
16	KUMARAN	38	MALE	+	-	+	+	AMBER	+	+	+	-	-	-	+	-	-	-	44	38	-	-	+	-	-	-	+	-	-	-	+	+	-	-	+	-	+	-	-	-	-
17	RAVIKUMAR	53	MALE	-	+	+	-	DULL	-	+		-	-	-	+	-	-	-	40	30	-	+	-	-	-	-	+	-	-	-	+	+	-	+	-	-	-	-	-	-	-
18	VELUSAMY	35	MALE	+	+	+	-	AMBER	+	-	+	-	-	-	+	-	-	-	58	17	-	-	+	-	-	-	+	-	-	-	+	-	-	-	-	-	-	-	+	-	+	-
19	MOHAMMED	10	MALE	+	-	-	-	AMBER	+	-	+	-	-	-	-	+	-	+	27	30	-	+	-	-	-	-	+	-	-	-	+	-	-	-	-	-	-	-	+	-	+	+
20	SUJITHA	9	FEMALE	+	-	+	-	AMBER	-	-	+	-	-	-	-	+	-	-	38	32	-	-	+	-	-	-	+	-	-	-	+	-	-	-	-	-	-	-	+	-	-	-
21	YASMINE	8	FEMALE	+	+	+	+	AMBER	-	-	-	-	-	-	-	+	-	+	40	44	-	+	-	-	-	-	+	-	-	-	+	-	+	-	-	-	-	-	+	-	-	-
22	RAJESH	28	MALE	-	+	+	-	AMBER	-	-	+	-	-	-	+	-	-	-	48	46	-	+	-	-	-	-	+	-	-	-	+	-	+	-	+	-	-	+	-	+	-	+
23	EVAJANLINE	7	FEMALE	-	+	+	-	DULL	+	-	-	-	-	-	-	+	-	+	36	30	-	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	+	-	+
24	KUMAR	10	MALE	+	-	-	-	AMBER	-	-	-	-	-	-	-	+	-	+	38	40	-	+	-	-	-	-	+	-	-	-	+	-	-	-	+	-	-	-	-	-	-	-
25	THAMESH	6	MALE	-	+	+	-	AMBER	-	-	+	-	-	-	-	+	-	+	30	37	-	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+
26	DHINESH	11	MALE	-	-	+	-	DULL	-	-	+	-	-	-	-	-	-	-	20	38	-	+	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	+	-	+	-
27	SREJITH	8	MALE	-	+	+	-	DULL	+	-	-	-	-	-	-	-	-	-	30	36	-	-	+	-	-	-	+	-	-	-	+	-	-	-	-	-	-	-	+	-	+	+
28	FAHAD	14	MALE	+	-	-	-	DULL	-	-	-	-	-	-	-	-	-	-	28	34	-	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	-	-	+
29	RAMESH	10	MALE	+	+	-	+	AMBER	+	-	+	-	-	-	-	+	-	+	40	42	-	-	+	-	-	-	+	-	-	-	+	-	-	-	-	-	-	-	+	-	+	+
30	VIJAY	8	MALE	-	+	+	-	AMBER	+	-	-	-	-	-	-	+	-	+	38	36	-	+	-	-	-	-	+	-	-	-	+	-	-	-	-	+	-	-	+	+	+	-
31	SHANTHI	9	FEMALE	+	-	-	+	AMBER	+	-	-	-	-	-	-	+	-	+	42	38	-	+	-	-	-	-	+	-	-	-	+	+	-	-	-	-	-	-	-	-	-	-
32	PANKAJAM	42	FEMALE	+	-	+	-	AMBER	+	-	-	-	-	-	+	-	-	-	17	30	-	-	+	-	-	-	-	+	-	-	+	-	-	+	-	+	-	-	-	-	-	+
33	LAVANYA	8	FEMALE	+	-	+	+	AMBER	+	-	-	+	-	-	-	-	+	-	36	20	-	+	-	-	-	-	+	-	-	-	-	+	+	-	-	-	-	-	-	-	-	-
34	JASMINE	16	FEMALE	+	+	-	-	DULL	-	-	+	-	-	-	-	-	-	-	38	30	-	+	-	-	-	-	+	-	-	-	-	-	-	-	+	-	-	-	+	-	+	+
35	SHARMILA	8	FEMALE	+	-	-	-	DULL	-	-	-	-	-	-	-	+	-	+	30	28	-	-	+	-	-	-	-	+	-	-	+	-	-	-	-	+	-	-	-	-	-	+
36	RANI	12	FEMALE	+	+	-	-	DULL	-	-	+	-	-	-	-	+	-	+	17	40	-	+	-	-	-	-	+	-	-	-	-	+	+	+	-	-	-	-	-	-	-	-
37	MEENA	38	FEMALE	-	+	+	-	DULL	+	-	-	-	-	-	+	-	-	-	30	38	-	-	+	-	-	-	+	-	-	-	+	+	-	+	-	-	-	+	-	-	-	-
38	RAJA	18	MALE	+	-	+	+	DULL	-	-	+	-	-	-	+	-	-	-	32	42	-	+	-	-	-	-	+	-	-	-	-	+	-	+	-	-	+	-	-	-	-	+
39	SENTHIL	20	MALE	-	-	+	-	AMBER	-	-	-	-	-	-	+	-	-	-	44	38	-	+	-	-	-	-	+	-	-	-	+	+	-	-	-	-	+	-	-	-	-	+
40	GAYATHRI	16	FEMALE	-	+	+	-	AMBER	+	-	-	-	-	-	-	-	-	-	46	18	-	+	-	-	-	-	+	-	-	-	+	-	-	-	+	-	-	+	-	+	+	-
41	MUTHU	10	MALE	+	-	+	-	AMBER	-	-	+	-	-	-	-	+	-	-	30	38	-	+	-	-	-	-	+	-	-	-	+	-	-	-	+	-	+	-	-	-	-	+
42	SADHASIVAM	12	MALE	-	-	-	-	DULL	+	-	-	-	-	-	-	+	-	-	40	23	-	-	+	-	-	-	-	-	-	-	+	+	-	-	-	-	-	-	-	-	-	-
43	AMUTHAN	28	MALE	+	-	-	-	DULL	-	-	+	-	-	-	+	-	-	-	37	38	-	+	-	-	-	-	+	-	-	-	+	+	-	+	-	-	-	-	-	-	-	-
44	SARAVANAN	24	MALE	-	+	+	+	DULL	-	-	-	+	-	-	-	-	-	-	38	30	-	+	-	-	-	-	+	-	-	-	+	-	-	-	-	-	-	-	+	-	-	-
45	CHARLES	13	MALE	+	-	-	-	AMBER	-	-	-	-	-	-	-	+	-	+	53	17	-	+	-	-	-	-	+	-	-	-	+	-	-	-	-	+	-	-	-	-	-	-
46	ASHOK KUMAR	36	MALE	+	-	+	-	AMBER	+	-	+	-	-	-	+	-	-	-	45	40	-	+	-	-	-	-	+	-	-	-	+	+	-	-	-	+	+	-	-	+	-	-
47	MUTHUSAMY	22	MALE	+	-	-	+	DULL	-	-	+	+	-	-	-	-	-	-	38	40	-	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-
48	PRIYA	14	FEMALE	-	-	-	-	AMBER	-	-	+	-	-	-	-	+	-	+	38	20	-	-	+	-	-	-	-	+	-	-	+	-	-	-	-	+	-	-	-	+	-	+
49	SARASWATHI	40	FEMALE	+	+	-	-	DULL	+	-	+	-	-	-	+	-	-	-	23	17	-	+	-	-	-	-	+	-	-	-	+	+	-	+	-	-	+	-	-	-	-	-
50	RAMAN	48	MALE	+	+	-	+	DULL	+	-	-	-	-	-	+	-	-	-	38	30	-	+	-	-	-	+	+	-	-	-	+	+	-	-	-	-	-	-	-	-	-	-